OCT 10 1939 TULANE UNIVERSITY
SCHOOL OF MEDICINE LIBRARY

Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

50 WEST 50TH STREET, NEW YORK, N. Y.

DR. JAMES G. CARR, Chicago, *Editor*

DR. GILBERT H. MARQUARDT, Chicago, *Associate Editor*

Vol. VIII **October, 1939** **No. 10**

CHRONIC CARDIAC COMPRESSION PRODUCED BY CONTRACTURE OF EXTRINSIC SCARS ON THE HEART

(Pick's Disease, Constrictive Pericarditis)

Diseases of the heart can be divided into two general groups — (1) intrinsic heart lesions and (2) extrinsic heart lesions. Extrinsic lesions of the heart disturb function by compression, by angulation or by rotation of the heart. In my experience these disturbances occur as entities, i.e., a compressed heart is not also angulated or rotated and an angulated heart is not also compressed. Traction upon adjacent structures through adhesions to the heart is not included as a component of extrinsic cardiac lesions. In my experience it is doubtful whether traction in the long axis of the heart can disturb cardiac function. If this statement is correct, and there is good experimental and clinical evidence to support it, the hypothesis of Brauer and the operation of Brauer are no longer tenable. As a rule, an extrinsic lesion does not extend into the heart and become intrinsic. A heart with an extrinsic deformity is to be considered as a good heart that has become crippled by an outside factor. It follows from this that if the extrinsic factor can be removed by operation the heart becomes normal. This statement is essentially correct although I have seen a few exceptions to it, conditions in which scar tissue extended into the myocardium from the outside. Likewise it can be stated that intrinsic disease, as a rule, does not become extrinsic. The adhesions that develop in rheumatic heart disease or over a myocardial infarct, both originating from intrinsic lesions, do not disturb cardiac function. These statements originated by the author are based largely upon experiences in experimental heart work. They should help to remove the confusion that now exists in nomenclature, diagnosis and operation.

The compression scars are always extrinsic in origin. They never develop as a sequel or complication of an intrinsic lesion. They never originate from rheumatic heart disease. The infection finds entrance into the pericardium from the pharynx, lungs, pleura or mediastinum. In my series of operated cases, seven showed evidence of tuberculosis in the scars and twenty-nine did not show evidence of tuberculosis in the scar removed at operation. Two of this group of twenty-nine patients, however, had tuberculosis elsewhere without showing tubercles in the scar.

The triad of Beck has made the diagnosis simple and infallible. The syndrome has been greatly simplified by placing emphasis upon compression and by eliminating the old fallacy that the heart pulling upon adjacent structures through adhesions results in dilatation, hypertrophy and failure of the organ. That the compressed heart is entirely free from any of the assumed mal effects of pulling upon adhesions, even though adhesions are everywhere present, has been demonstrated beyond any reasonable doubt. The diagnostic triad consists of (1) a small quiet heart, (2) a high venous pressure in the arm and (3) ascites and a large liver. The diagnosis often can be made by inspection alone. The precordium is quiet. Under the fluoroscope, the pulsation is reduced in amplitude. The sounds are distant. There are no murmurs. One would not expect murmurs in one of the extrinsic forms of heart disease. The venous pressure in the arm may be as high as 42 cms. of physiological solution of sodium chloride. Usually this pressure measures from 20 to 32 cms. It indicates stasis in the superior vena cava. The enlarged liver and ascites indicate stasis in the inferior vena cava. Many manifestations of secondary importance can be mentioned. These are dilatation of the veins, cyanosis, increase in circulating blood volume, depletion of oxygen in the venous blood, reduction in the protein content of the blood, increase in the cerebro-spinal fluid pressure, increase in lymphatic and thoracic duct pressure, subcutaneous edema, hydrothorax, pulmonary edema, a narrow pulse pressure (systolic pressure commonly 100 mm. Hg., diastolic pressure 80 mm. Hg.), reduction in cardiac output, malnutrition and weakness. The muscle fibers in the compressed heart are definitely smaller than normal as shown in recent studies by Dr. Roberts and myself. The heart undergoes atrophy of disuse and requires weeks or months to regain its normal strength after resection of a compression scar. The size of the entire heart is smaller than normal. This was demonstrated in cardiac compression produced experimentally. That the heart gets larger after resection of the scar was demonstrated repeatedly in patients who were operated upon. The compressed heart is an efficient organ. It does not waste energy. It does not receive a normal quota of blood but what it receives is propelled by the heart without abnormal resistance or obstruction. The electrocardiogram shows a reduction in voltage. In some cases the heart is fixed to the diaphragm or sternum and in some cases it is not fixed. Fixation of the compressed heart is of no significance.

As a rule, cardiac compression does not assume any importance to the patient until ascites forms. In our experiments ascites was first noticed when the venous pressure rose to 15 cms. The lowest venous pressure in any of my patients with ascites was 20 cms. The venous pressure can be regarded as an accurate measurement of the pressure upon the heart. Pressures of 8 to 10 cms. on the heart are well tolerated if the pressure is built up slowly. An increase in circulating blood volume takes place in response to the compression and, this in turn, raises

venous pressure, which in turn, injects sufficient blood into the heart to maintain a satisfactory circulation. The venous pressure must always be higher than the compression forces on the heart. The truth of this statement is obvious and it can be considered as a physiological law. If the pressure upon the heart were greater than the pressure in the venae cavae blood could not enter the heart.

The only treatment for compression scars is operative. If the patient is febrile it is advisable to wait until the infection becomes quiescent. It may not become quiescent and the patient may become so emaciated that operation must be carried out in the presence of active infection. The patient is dehydrated in preparation for operation. When fluid is taken from the abdomen the venous pressure falls and the pressure differential, so essential for filling of the heart, is sometimes reduced to a dangerous level. To elevate the venous pressure and to insure cardiac filling, an abdominal binder is used as the fluid is removed. Avertin and other basal anesthetic agents should not be used because, with these drugs, respiration might stop. We use a small dose of morphine and nitrous oxide anesthesia. The anesthetic should be light and must be carefully given. The incision that I use is transverse below the left nipple. The pectoral muscle is exposed and is cut across its fibers about 1 cm. from its origin. The 4th and 5th, and rarely the 3rd or 6th, costal cartilages are completely removed. The left pleural sinus is dissected from the scar. The scar is incised from apex to base and is separated from the heart as extensively as possible. It should be incised well up over the base of the heart. The separation may be easy or difficult. It may require sharp dissection. As much of the scar is excised as can be safely done. The scar posterior to the heart does not need to be taken away. The things to be avoided are pressure upon a coronary artery, cutting a coronary artery, tearing the ventricle or auricle and dislocations of the heart. If the heart is irritable, rapid or irregular, rest periods are necessary. Procaine applied locally can be useful if the heart is responsive to surgical stimuli from its surface. Anoxemia of the heart is dangerous and should be corrected if possible. The heart dilates after the compression is released. One likes to see this because it indicates release of compression. At the same time if the dilatation is extreme the heart might stop. At this point the anesthetist must be careful not to blow up the lungs forcibly because the pulmonary resistance is thereby increased and the right ventricle dilates further. Reduction of venous pressure by venesection might be indicated from this point on. I have not done venesection but I shall if indicated. The left pleural cavity is opened to provide internal drainage. After operation fluid is aspirated from the pleural cavity as necessary. Drainage to the outside is always contra-indicated. The pectoral muscle is sutured in place. Air is sucked from the mediastinum and pleural cavity through a catheter while closure is carried out. Schmieden, Sauerbruch, Winkelbauer, Denk, Westermann, Volhard and Fischer recommend removal of the pectoral muscle and the bony precordium so that when the heart becomes adherent to the chest wall, it will have only skin to pull upon. This is an attempt to combine the Brauer operation with the removal of the compression scar. These authors believe the pull upon adhesions is a factor in this condition. Dilatation and hypertrophy of the heart, which are supposed to result from adhesions, are thus combined in the same organ with the atrophy of compression. The excellent results that can be obtained without removing the pectoral muscle and bony precordium indicate to me that this paradoxical belief must be fallacious. The same statement can be made concerning the suggestion of Payne, Sauerbruch, Fischer and Schmieden, also other German authors, that the left diaphragm be paralyzed by cutting the phrenic nerve. Complete recovery can be obtained without paralysis of the diaphragm. After operation the patient is placed in semi-Fowler's position in an oxygen tent. Fluid is not given except as requested by the patient. Tachycardia and irregularities in the pulse are treated by digitalis and quinidine.

Improvement is always noted during and after operation. Sometimes the early improvement is lost after several hours because of the dilated, atrophic condition of the heart. Usually the patient feels better as soon as he becomes conscious. He breathes more easily. His hands and feet are warmer than they were before operation. The veins are less distended. Diuresis sometimes begins early. Sometimes it is delayed weeks or months after operation until the heart recovers from its atrophic condition. Results: Analysis of the literature shows that 175 patients with compression scars were operated upon by 51 surgeons. Of these Beck had 36 (1939), Schmieden 22 (1937), Bialock 14 (1939), Churchill 12 (1936), Tengwall 7 (1938), Heuer 6 (1938), L. Rehn 4 (1920), DeQuervain 4 (1934), Sauerbruch 4 (1925), Borchardt 4 (1938), Lawen 3 (1928), Volker 3 (1933), Whittemore 3 (1935), Budisavljevic 3 (1931). The number of patients listed as cured was 73 or 41.7 per cent, as improved 41 or 23.4 per cent, as not improved 1 or 0.6 per cent, as not classified 3 or 1.7 per cent, and as died 57 or 32.6 per cent. It should be pointed out that the majority of these patients can be completely cured by operation. Of my series of 36 patients operated upon, 23 are completely free of ascites and edema and can be classified as cured; 3 additional cases have been operated upon recently and probably will be cured; 3 patients were improved and 7 died. Of the 3 patients improved, 1 has invasion of the myocardium by scar, 1 had reformation of calcium deposits around and in the myocardium and 1 had an incomplete operation (a second operation should be done on this patient). Of the 7 deaths, 3 had active tuberculosis in the pleurae and mediastinum, 1 had an epicardial scar plus bloody fluid and died of infection, 2 died of heart failure from atrophy of the heart, and 1 of ventricular fibrillation during operation. At the present time we are prepared to defibrillate the ventricles at the time of operation. It might be mentioned that recently the author successfully defibrillated the human ventricles. This is the first time this has been accomplished.

Claude S. Beck, M.D.
Cleveland, Ohio